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Abstract: Objectives: Controversy still exists regarding the optimal surgical technique for postinfarction dyskinetic left ventricular aneurysm (LVA) repair. We compared the efficacy of two established techniques, linear vs. patch remodeling, for repair of dyskinetic LVA. Patients and methods: From 1989 to 1998, 95 (16 women, 79 men) consecutive patients were operated on for postinfarction dyskinetic LVA. Thirty-four patients underwent patch remodeling (R) and 61 linear (L) repair. The mean age was 61.1±8.5 years. Indications for surgery alone or in combination included angina in 72 patients, dyspnea in 64 and ventricular tachycardia in 41. Thirty-seven patients had a history of congestive heart failure (R 13 (38%), L 24 (39%), NS). The mean ejection fraction (EF) with aneurysm was 0.29±0.09 in R vs. 0.35±0.10 in L (P 0.04), whereas the mean EF without aneurysm was 0.43±0.11 in R vs. 0.46±0.08 in L (P=0.3). Seventy-one aneurysms were anterior (R 30 (88%), L 41 (68%), P 0.05). Concomitant coronary artery bypass grafting was performed in 84 patients (R 29 (85%), L 55 (90%), NS). Follow-up ranged from 1 to 12 years (mean 5.6±3.4 years, median 6.1 years). Results: Early mortality was 8% (n=8) (R 4, L 4, NS). Survival at 1, 5 and 10 years was 88, 73, and 44%, respectively. It did not differ significantly between R (1 and 5 year survival 85, 66%) and L (90, 76%, P=0.58). Preoperative risk factors for mortality were history of congestive heart failure (1 and 5 year survival 81 and 57% vs. 90 and 78%, respectively, hazard ratio (HR)=1.95, P 0.05), non-anterior localization of the aneurysm (86 and 49% vs. 86 and 77%, HR=2.06, P 0.05), history of thromboembolic events (57 and 19% vs. 89 and 74%, HR=3.27, P 0.05), and left ventricular EF (HR=0.97 per %, P=0.05). At late follow-up the mean functional class was 1.8±0.6 in long-term survivors (preoperative 2.9±0.9, P 0.001) with no difference between the groups. Conclusions: The technique of repair of postinfarction dyskinetic LVA should be adapted in each patient to the cavity size and extent of the scarring process into the septum and subvalvular mitral apparatus. Applying these considerations to the choice of the technique of repair, both techniques achieved satisfactory results with respect to perioperative mortality, late functional status and survival

DOI: https://doi.org/10.1016/s1010-7940(02)00210-5

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ELSEVIER

European Journal of Cardio-thoracic Surgery 22 (2002) 129–134

EUROPEAN JOURNAL OF CARDIO-THORACIC SURGERY

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Repair of postinfarction dyskinetic LV aneurysm with either linear or patch technique ☆

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Received 13 September 2001; received in revised form 7 March 2002; accepted 25 March 2002

Abstract

Objectives: Controversy still exists regarding the optimal surgical technique for postinfarction dyskinetic left ventricular aneurysm (LVA) repair. We compared the efficacy of two established techniques, linear vs. patch remodeling, for repair of dyskinetic LVA. **Patients and methods**: From 1989 to 1998, 95 (16 women, 79 men) consecutive patients were operated on for postinfarction dyskinetic LVA. Thirty-four patients underwent patch remodeling (R) and 61 linear (L) repair. The mean age was 61.1 ± 8.5 years. Indications for surgery alone or in combination included angina in 72 patients, dyspnea in 64 and ventricular tachycardia in 41. Thirty-seven patients had a history of congestive heart failure (R 13 (38%), L 24 (39%), NS). The mean ejection fraction (EF) with aneurysm was 0.29 ± 0.09 in R vs. 0.35 ± 0.10 in L ($P < 0.04$), whereas the mean EF without aneurysm was 0.43 ± 0.11 in R vs. 0.46 ± 0.08 in L ($P = 0.3$). Seventy-one aneurysms were anterior (R 30 (88%), L 41 (68%), $P < 0.05$). Concomitant coronary artery bypass grafting was performed in 84 patients (R 29 (85%), L 55 (90%), NS). Follow-up ranged from 1 to 12 years (mean 5.6 ± 3.4 years, median 6.1 years). **Results**: Early mortality was 8% ($n = 8$) (R 4, L 4, NS). Survival at 1, 5 and 10 years was 88, 73, and 44%, respectively. It did not differ significantly between R (1 and 5 year survival 85, 66%) and L (90, 76%, $P = 0.58$). Preoperative risk factors for mortality were history of congestive heart failure (1 and 5 year survival 81 and 57% vs. 90 and 78%, respectively, hazard ratio (HR) = 1.95, $P < 0.05$), non-anterior localization of the aneurysm (86 and 49% vs. 86 and 77%, HR = 2.06, $P < 0.05$), history of thromboembolic events (57 and 19% vs. 89 and 74%, HR = 3.27, $P < 0.05$), and left ventricular EF (HR = 0.97 per %, $P = 0.05$). At late follow-up the mean functional class was 1.8 ± 0.6 in long-term survivors (preoperative 2.9 ± 0.9, $P < 0.001$) with no difference between the groups. **Conclusions**: The technique of repair of postinfarction dyskinetic LVA should be adapted in each patient to the cavity size and extent of the scarring process into the septum and subvalvular mitral apparatus. Applying these considerations to the choice of the technique of repair, both techniques achieved satisfactory results with respect to perioperative mortality, late functional status and survival.

Keywords: Postinfarction; Dyskinetic; Left ventricular aneurysm; Repair

1. Introduction

Transmural myocardial infarction may be complicated by formation of a left ventricular aneurysm (LVA). Postinfarction dyskinetic LVA distorts the normal elliptical geometry of the left ventricle, to produce a dilated spherical ventricle with limited contractile and filling capacities [1]. The consequences are congestive heart failure, thromboembolic complications, associated arrhythmias, repeated hospitalizations, and eventually death.

Surgical repair of LVA was first performed by Charles Bailey in 1954 [2]. The first resection under cardiopulmonary bypass was reported by Denton Cooley and associates in 1958 [3]. During the following decades besides the traditional linear repair, newer patch remodeling techniques have been conceived and reported in an effort to improve results [4–6]. However, controversy still exists about the optimal repair technique for postinfarction dyskinetic LVA. This retrospective study evaluates the two techniques of repair of postinfarction dyskinetic LVA, i.e. patch remodeling and linear repair, by assessment of early and late

☆ Presented at the joint 15th Annual Meeting of the European Association for Cardio-thoracic Surgery and the 9th Annual Meeting of the European Society of Thoracic Surgeons, Lisbon, Portugal, September 16–19, 2001.

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1010-7940/02/$ - see front matter
PII: S1010-7940(02)00210-5

outcome of patients undergoing surgery for postinfarction dyskinetic LVA.

2. Patients and methods

2.1. Definitions

Dyskinetic LVA was defined as a segment of left ventricular wall protruding from the expected outline of the ventricular chamber, displaying paradoxical motion on left ventriculogram [7]. In all patients, dyskinetic LVA was confirmed at operation.

Paired pre- and postoperative echocardiographic examinations were available in 39 patients (patch $n = 12$, linear $n = 27$), consisting of four standard views (parasternal, long axis, short axis at the papillary muscle level and apical two- and four-chamber). Standard measurements were obtained for septal and posterior left ventricular wall thickness, left ventricular end-systolic volume (LVESV) and end-diastolic volume (LVEDV) dimensions. Three measurements were averaged to determine ejection fractions (EFs) calculated in the four-chamber view with the modified Simpson's rule. EF was calculated with and without inclusion of the aneurysm. Pulsed Doppler echocardiography was performed to determine the presence and severity of mitral regurgitation.

Coronary angiography was performed in all patients. Coronary stenosis was considered significant if there was a 70% or greater stenosis in the luminal diameter in any view. A stenosis of 50% or more in the left main coronary artery was considered significant [8]. Left ventricular function was assessed by ventriculography or echocardiography, or both, and categorized as normal (EF > 0.5) in seven patients (seven linear), moderately impaired (EF $= 0.3$–0.5) in 58 (20 patch, 38 linear) or severely impaired (EF < 0.3) in 28 (13 patch, 15 linear) [9]. In two patients data relating to EF were missing. The mean EF with aneurysm was 0.29 ± 0.09 in patch remodeling (R) vs. 0.35 ± 0.10 in linear repair (L) ($P < 0.04$), whereas the mean EF without aneurysm was 0.43 ± 0.11 in R vs. 0.46 ± 0.08 in L ($P = 0.3$). Three-vessel disease was present in 55 patients (patch 18 (53%), linear 37 (61%), NS), and left main disease was present in five patients (patch 1 (3%), linear 4 (6.5%), NS). LAD disease was found in 90 patients (patch 32 (94%), linear 58 (95%), NS). Seventy-one aneurysms were anterior (patch 30 (88%), linear 41 (68%), $P < 0.05$).

2.2. Patient characteristics

From January 1989 through December 1998, 95 consecutive patients underwent surgical repair of postinfarction dyskinetic LVA. Thirty-four patients underwent patch remodeling and 61 linear repair according to the surgeon's preference.

There were 79 men (26 patch, 53 linear, NS) and 16 women (8 patch, 8 linear, NS) (Table 1). The mean age was 61.1 ± 8.5 years (patch 61.8 ± 8.7, linear 60.8 ± 8.4, NS). Seventy-one patients were in New York Heart Association (NYHA) functional class III or IV (patch 24 (71%), linear 47 (77%), NS). A history of congestive heart failure was present in 37 patients (patch 13 (38%), linear 24 (39%), NS). Preoperatively, 72 patients presented with angina (patch 20 (65%), linear 50 (82%), NS), 64 with dyspnea (patch 25 (75%), linear 39 (64%), NS), 41 with ventricular tachycardia (patch 17 (50%), linear 24 (39%), NS) and eight with a history of an embolic event (patch 2 (6%), linear 6 (10%), NS). Twenty patients were survivors of sudden cardiac death (patch 7 (21%), linear 13 (21%), NS).

Table 1
Preoperative hemodynamics

	Patch	Linear	P value
Mean pulmonary artery pressure (mmHg)	18 ± 6	16 ± 7	NS
Pulmonary wedge pressure (mmHg)	10 ± 5	10 ± 6	NS
EF with aneurysm	0.29 ± 0.09	0.35 ± 0.10	<0.04
EF without aneurysm	0.43 ± 0.11	0.46 ± 0.08	NS
Diseased LV circumference (%)	39 ± 4	38 ± 6	NS

Two patients (2 linear) had undergone previous open heart surgery. Four patients (4 linear) had $\geq +3$ mitral regurgitation. Ten patients (3 patch, 7 linear) were operated on as emergencies. Preoperative hemodynamic data are reported in Table 1. Preoperatively left ventricular volumes were higher in the linear group as compared to those of the patch group (Figs. 1 and 2) but the difference did not reach statistical significance ($P = 0.07$ for end-diastolic and $P = 0.1$ for end-systolic volumes).

2.3. Surgical technique

All surgical procedures were performed by a total of six surgeons. Cardiopulmonary bypass was conducted using moderate hypothermia and antegrade or combined antegrade and retrograde blood cardioplegia. Nine patients had

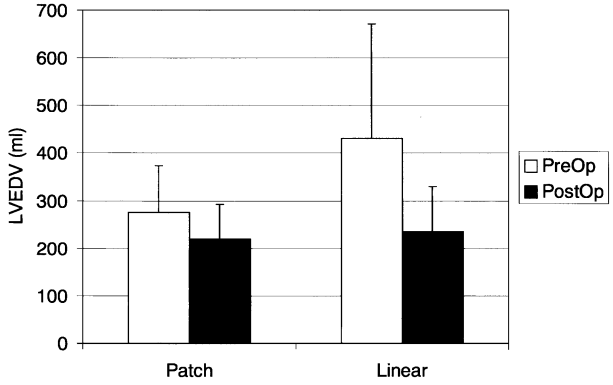

Fig. 1. Left ventricular end-diastolic volumes (LVEDV) before and early after operation. Baseline LVEDV tended to be larger in the linear repair group ($P = 0.07$).

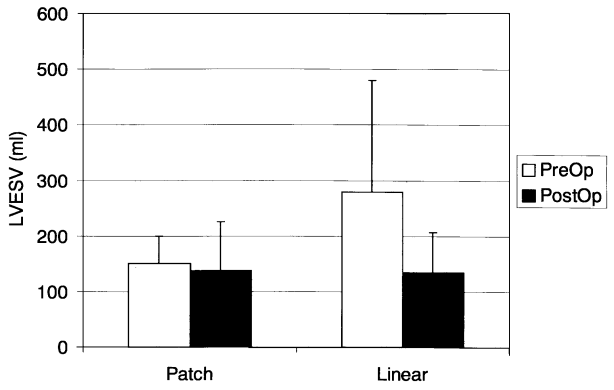

Fig. 2. Left ventricular end-systolic volumes (LVESV) before and early after operation. Baseline LVESV tended to be larger in the linear repair group ($P = 0.1$).

their operation performed using induced ventricular fibrillation. Under cardiopulmonary bypass the diagnosis of dyskinetic LVA was confirmed visually and by palpation of the thinned wall of the left ventricle. Insertion of a left ventricular vent was avoided before the aorta was clamped or the ventricular fibrillation induced. Coronary artery bypass grafting was done as indicated and additional cardioplegia was routed through the grafts in the arrested hearts. The left ventricle was then opened over the thinned wall and clots if present were removed. Depending on the size and shape of the left ventricular cavity a portion of the thinned wall was resected. For patients with malignant ventricular tachycardia non-guided cryoablation and subendocardial resection were performed as indicated. Two types of aneurysm closure, patch [3–5] or linear, were used depending on the personal preference of each surgeon and on the size of the left ventricular cavity.

2.4. Follow-up data

Follow-up data were obtained directly from the patients and/or their general practitioners by telephone calls or written questionnaire. Long-term follow-up was missing in seven patients (7%). Follow-up was censored in these patients at the time of last visit in our hospital. The duration of follow-up for hospital survivors ranged from 1 to 12 years. The mean follow-up for the whole study population was 5.6 ± 3.4 years (median 6.1 years).

2.5. Statistical analysis

Continuous variables are expressed as the mean ± 1 SD. Categorical variables are presented as percentages. Groups were compared with use of Fischer's exact test, Student's *t*-test or the Mann–Whitney *U*-test, as appropriate. Survival curves are presented according to the method of Kaplan and Meier. The significance of each variable as a prognostic marker was first tested univariately using a log-rank test for categorical variables and the Cox proportional hazards model for continuous variables. Variables identified as significantly associated with outcome were examined multivariately using the stepwise Cox proportional hazards model. Differences of $P < 0.05$ were considered to be statistically significant.

3. Results

3.1. Intraoperative variables

The mean cross-clamp time was 34 ± 23 min (patch 31 ± 30 min, linear 37 ± 19 min, NS), and the mean cardiopulmonary bypass time was 93 ± 41 min (patch 105 ± 44 min, linear 86 ± 38 min, $P < 0.05$). Concomitant coronary artery bypass grafting was done in 84 patients (patch 29 (85%), linear 55 (90%), NS). Revascularization was complete in 36 patients (patch 10 (29%), linear 26 (43%), NS). The left anterior descending artery was revascularized in 44/90 patients with LAD disease (49%) (patch 11/32 (34%), linear 33/58 (57%), $P < 0.05$), 31 with the left internal mammary artery (patch 9/11 (81%), linear 22/33 (66%), NS). The average distal anastomoses/patient was 2.3 ± 1.5 (patch 2.2 ± 1.6, linear 2.3 ± 1.4, NS).

Subendocardial resection was undertaken in 27 patients and cryoablation in 13.

Mitral valve replacement was done in two patients with posterior chordal preservation using bileaflet mechanical valves, and mitral valve repair in another two patients.

3.2. Outcome

There were eight in-hospital deaths (8%). The causes of hospital deaths were pump failure in five patients, cerebrovascular insult in two and sepsis in one. There was no significant difference in early mortality between the patch and linear groups (patch 4/34 (12%), linear 4/61 (7%), NS). No predictor of early mortality could be identified.

Perioperative morbidity included intraaortic balloon pumping in 17 patients (18%; 10 inserted preoperatively), respiratory support for more than 24 h in 48 patients (51%), reexploration for bleeding in six (6%), cerebrovascular insult in six (6%), myocardial infarction in two (2%), mediastinitis in three (3%) and need for implantable cardiovertor defibrillator in three (3%). There was no significant difference in early morbidity between the patch and linear groups. Postoperative hemodynamic data are reported in Table 2.

Table 2
Postoperative hemodynamics

	Patch	Linear	*P* value
Mean pulmonary artery pressure (mmHg)	19 ± 7	16 ± 6	NS
Pulmonary wedge pressure (mmHg)	10 ± 8	8 ± 5	NS
EF	0.44 ± 0.11	0.45 ± 0.09	NS

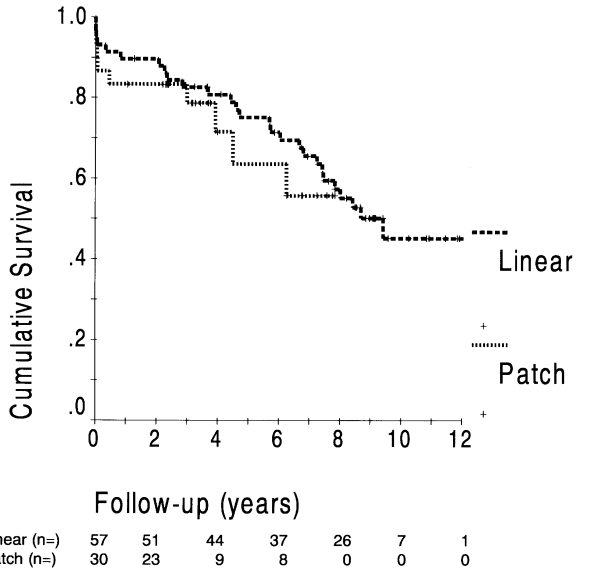

Fig. 3. Cumulative survival rates for patients with patch or linear repair, showing no significant difference between the two groups. Numbers of patients at risk at each interval are indicated at the bottom.

Postoperatively both LVEDV (Fig. 1) and LVESV (Fig. 2) decreased to the same extent in both groups.

Late morbidity included cerebrovascular insults in nine patients (four due to thromboembolic events of cardiac origin), myocardial infarction in three (two of which were fatal), bleeding in one and need for cardiovertor-defibrillator implantation in six. Late death occurred in 28 patients, with 16 cardiac-related. The other causes of death were cancer in five patients, pneumonia in one and unknown in six. The actuarial survival for the entire group including both perioperative and long-term survival is presented in Fig. 3. Survival rates at 1, 5 and 10 years were 88, 73 and 44%, respectively. There was no significant difference in late survival between the patch and linear groups. In univariate analysis history of congestive heart failure (hazard ratio (HR) = 2.18, $P = 0.02$), history of thromboembolic event (HR = 2.91, $P = 0.03$), non-anterior location of myocardial infarction (HR = 1.99, $P = 0.05$) and EF (HR = 0.96 per %, $P = 0.03$) were found to be associated with increased

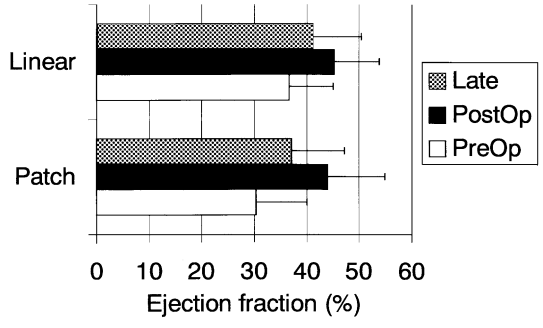

Fig. 4. The baseline EF (with aneurysm) was lower in patients with patch repair ($P < 0.04$). The early postoperative increase was significant in both groups ($P < 0.01$) and did not differ significantly between the groups.

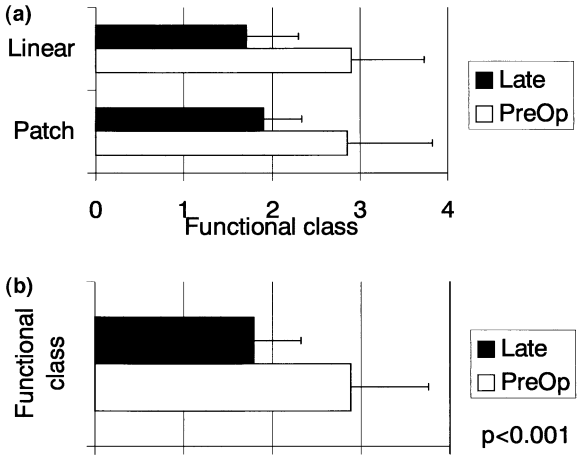

Fig. 5. (a,b) Functional class according to the NYHA before and late after operation in patients with patch and linear repair. The functional improvement was statistically significant in both groups ($P < 0.001$), but did not differ between the groups.

overall mortality. Multivariate analysis identified EF (HR = 0.955 per %, $P = 0.007$), non-anterior location of myocardial infarction (HR = 2.20, $P = 0.04$) and history of thromboembolic event (HR = 4.95, $P = 0.003$) as independent risk factors for overall mortality.

The EF as measured by echocardiography improved to the same degree in both groups (NS between groups; Fig. 4). The improvement in the EF was still present late after the operation in both groups. The functional class improved in hospital survivors from 2.9 ± 0.9 preoperatively to 1.8 ± 0.5 at the last follow-up ($P < 0.001$). There was no difference between the groups (patch from 2.9 ± 1.0 to 1.9 ± 0.4, linear from 2.9 ± 0.8 to 1.7 ± 0.6, NS) (Fig. 5a,b).

4. Discussion

Resection of dyskinetic LVA has been an accepted treatment in patients with postinfarction congestive heart failure, thromboembolic complications or significant ventricular arrhythmias. The linear closure of the ventriculotomy introduced in 1958 by Cooley and coworkers [3] and its various modifications [10,11] has remained the technique of choice in most instances [12–15]. On the basis of theoretic considerations [16–18] patch remodeling of postinfarction LVA has been advocated [19,20] to improve ventricular geometry and thereby early and late outcome. Retrospective clinical studies have opposed linear to patch repair and reported varying results [12,21]. In these studies, the role of concomitant coronary artery bypass grafting has been overlooked although it has been shown that myocardial revascularization improves the long-term survival in the high-risk subgroups of patients with LVA [7].

In this study we compared the early and late outcome of consecutive patients undergoing repair of postinfarction

dyskinetic LVA with either linear or patch remodeling techniques. Both techniques were used simultaneously during the study period and there was no difference in the intraoperative management of the patients with respect to cardiopulmonary bypass and myocardial protection strategy. Also, there was no difference between the two groups regarding the extent and the completeness of myocardial revascularization. Although the cardiopulmonary bypass time was slightly longer in the patch repair group, this did not affect the results on outcome. Preoperatively the EF including the aneurysm was lower in the patch group ($P < 0.04$). However, after excluding the aneurysm from the calculation of the EF the difference between the groups lost its statistical significance ($P = 0.3$). Conversely more patients in the linear group had posteroinferior aneurysm, which was an independent risk factor for overall mortality. All other parameters including the extent of coronary artery disease were similar between the two groups. The choice of the surgical procedure depended on the preference of the surgeon guided by the size of the left ventricular cavity and on the degree of involvement of the interventricular septum and subvalvular mitral apparatus. This was reflected in the larger preoperative volumes of patients who underwent linear compared to patch techniques. Left ventricular volumes decreased postoperatively to the same extent in both groups. The overall operative mortality was 8% which compares with the reports of other authors [7,11,13,14]. It was not different between the two groups. The EF as well as the late functional status improved significantly and to the same extent in both groups. This improvement in EF and functional status is similar to that reported in other series [13,15,22,23]. Again regarding the improvement in EF and functional status there was no significant difference between the linear and patch groups. Multivariate analysis identified preoperative EF, non-anterior location of myocardial infarction and history of thromboembolic event as independent risk factors for overall mortality. Preoperative EF has already been reported to adversely affect the overall mortality [11]. Importantly, long-term survival was not different between the linear and patch groups. The overall survival rates were 88, 73 and 44% at 1, 5 and 10 years and compare with those reported by other authors [7,11,13–15].

Our attempts at restoring left ventricular geometry to its conical shape during repair of postinfarction LVA consisted of alternatively using the linear or the patch repair for closing the ventriculotomy. The linear repair was preferably applied to patients with larger preoperative left ventricular volumes. In less enlarged ventricles extensive resection and linear closure of the ventriculotomy could compromise the diastolic function of the left ventricle. In this situation of restricted ventricular stroke volume, cardiac output becomes a function of pulse rate, and may result in low cardiac output and pump failure. Other draw-backs of the linear repair are that it cannot be satisfactorily applied in patients with extensive fibrosis of the interventricular septum, or in patients with a scarring process extending to the subvalvular mitral apparatus. In these cases, we believe that reconstruction of the left ventricular cavity with a patch is preferable. We also agree with Dor and coworkers [20] that patch repair is the more valuable technique in patients with severely impaired left ventricular function. Bearing in mind these considerations, we found no difference in early and late outcome of patients undergoing repair of postinfarction dyskinetic LVA with either linear or patch techniques. We believe that the technique of repair of postinfarction dyskinetic LVA should be adapted in each patient to these anatomical and physiological considerations. Moreover, revascularization of diseased vessels should be performed, whenever possible, in an attempt to improve late survival especially in patients with multivessel coronary artery disease [20].

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